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Delaying the onset of Alzheimer disease

Bilingualism as a form of cognitive reserve

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ABSTRACT

Objectives: There is strong epidemiologic evidence to suggest that older adults who maintain an active lifestyle in terms of social, mental, and physical engagement are protected to some degree against the onset of dementia. Such factors are said to contribute to cognitive reserve, which acts to compensate for the accumulation of amyloid and other brain pathologies. We present evidence that lifelong bilingualism is a further factor contributing to cognitive reserve.

Methods: Data were collected from 211 consecutive patients diagnosed with probable Alzheimer disease (AD). Patients' age at onset of cognitive impairment was recorded, as was information on occupational history, education, and language history, including fluency in English and any other languages. Following this procedure, 102 patients were classified as bilingual and 109 as monolingual.

Results: We found that the bilingual patients had been diagnosed 4.3 years later and had reported the onset of symptoms 5.1 years later than the monolingual patients. The groups were equivalent on measures of cognitive and occupational level, there was no apparent effect of immigration status, and the monolingual patients had received more formal education. There were no gender differences.

Conclusions: The present data confirm results from an earlier study, and thus we conclude that lifelong bilingualism confers protection against the onset of AD. The effect does not appear to be attributable to such possible confounding factors as education, occupational status, or immigration. Bilingualism thus appears to contribute to cognitive reserve, which acts to compensate for the effects of accumulated neuropathology. *Neurology*® 2010;75:1726-1729

GLOSSARY

AD = Alzheimer disease; MMSE = Mini-Mental State Examination.

Although evidence has accumulated validating the role of cognitive reserve in protecting against age-related dementias,¹ the relation between such factors as education and enhanced cognitive reserve is necessarily correlational. That is, it is unclear whether intellectual, social, and physical activities genuinely improve cognitive performance, or whether individuals with better-functioning brains (perhaps for genetic reasons) are more likely to perform well intellectually and are also protected naturally against the onset of dementia. This is not the case for bilingualism: in the vast majority of cases people become bilingual not because they are naturally gifted language learners, but because of circumstances that require it. The possibility that bilingualism may contribute to cognitive reserve and thus be associated with a delayed onset of dementia was suggested by a series of studies showing that the constant use of 2 or more languages enhances aspects of attention and cognitive control across the lifespan.²⁻⁴

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This suggestion was explored in a previous study of hospital records of 184 patients diagnosed with dementia, of whom 91 were monolingual and 93 were bilingual.⁵ Of these, 72% had been diagnosed with probable Alzheimer disease (AD) and the remainder with other forms of dementia. At the time of the first clinic appointment, the 2 language groups did not differ in scores on the Mini-Mental State Examination (MMSE) or occupational status, but the monolingual group had received more education (12.4 years) than the bilingual group (10.8 years), a difference that should favor monolingual subjects.⁶ However, the results showed that the estimated ages at onset of dementia were 71.4 years for monolingual subjects and 75.5 years for bilingual subjects; ages at first clinic appointment were 75.4 years for monolingual subjects and 78.6 years for bilingual subjects. Thus, bilingual patients exhibited symptoms of dementia between 3 and 4 years later than a comparable group of monolingual patients.

Here, we followed up this result with a new sample of patients. The need for a replication was underlined by the results of a recent study⁷ that found protective effects of multilingualism in some but not all groups. In the present study, we collected more detailed language information through a structured questionnaire and restricted the study to patients diagnosed with probable AD.

METHODS **Study design and patient selection.** We report data from 211 consecutive patients diagnosed with prob-

able AD in the Sam and Ida Ross Memory Clinic at Baycrest in Toronto, Canada, between January 2007 and December 2009. Diagnosis was based upon National Institute of Neurological and Communicative Disorders and Stroke–Alzheimer’s Disease and Related Disorders Association criteria⁸ and was reached by consensus among a group comprising at least 2 physicians and 1 neuropsychologist. At the first clinic visit, age at onset of cognitive impairment was recorded from patients or their caregivers. Information was also collected about occupational history, education, language history, fluency in English and other languages, place of birth, and date of immigration to Canada. The criterion for classification as bilingual was having spent the majority of life, at least from early adulthood, regularly using at least 2 languages. Accordingly, 102 patients were classified as bilingual (60 female) and 109 as monolingual (60 female). The bilingual subjects included speakers of 21 first languages, of which the most common were Yiddish (n = 24), Polish (n = 12), Italian (n = 11), Hungarian (n = 9), and French (n = 7).

Standard protocol approvals, registrations, and patient consents. This study received approval from the Baycrest Research Ethics Board in a Certificate of Research Approval dated August 11, 2006 (REB #06-41).

RESULTS The results are shown in the table. A 2-way analysis of variance for language group and gender showed that the 5.1-year difference in age at onset of symptoms of dementia yielded $F_{1,205} = 16.25$, $p < 0.0001$, with no other effects, $F_s < 1$. Estimation of onset age is clearly subjective, but we see no reason for a systematic difference between language groups in this respect. In any case, the 4.3-year difference in age for the initial clinic visit yielded $F_{1,207} = 12.02$, $p < 0.0006$, again with no other effects, $F_s < 1$. Given that the bilingual group contained more immigrants than the monolingual group, it might have been the case that patients in the bilingual group waited longer before consulting a doctor, but the duration between symptom onset and clinic visit was longer for monolingual subjects (3.8 years) than bilingual subjects (3.1 years),

Table		Mean value (SD) for descriptors for each language group					
Language group	No.	Age at onset, y ^a	Age at first appointment, y ^b	Duration, y ^c	MMSE ^d at first appointment	Years of education	Occupation status ^e
Monolingual	109	72.6 (10.0)	76.5 (10.0)	3.8 (2.9)	21.5 (5.7)	12.6 (4.1)	2.8 (1.3)
Men	49	73.3 (9.4)	77.3 (8.9)	3.9 (2.9)	22.1 (5.7)	13.2 (4.4)	3.2 (1.0)
Women	60	72.1 (10.4)	75.9 (10.8)	3.7 (2.9)	21.0 (5.7)	12.0 (3.8)	2.5 (1.3)
Bilingual	102	77.7 (7.9)	80.8 (7.7)	3.1 (1.9)	20.4 (5.6)	10.6 (5.1)	2.5 (1.1)
Men	42	77.6 (7.8)	80.4 (7.8)	2.8 (1.8)	21.0 (4.8)	11.1 (6.1)	3.0 (0.9)
Women	60	77.8 (8.1)	81.1 (7.6)	3.3 (1.9)	20.0 (6.0)	10.3 (4.3)	2.2 (1.2)

Abbreviation: MMSE = Mini-Mental State Examination.

^a Age at which symptoms were first reported by family.

^b Age at first visit to clinic.

^c Duration of elapsed time between ^a and ^b.

^d Scores out of 30.

^e Based on 4-point scale developed by Human Resources and Skills Development, Canada, in which higher numbers signify higher status.

$F_{1,205} = 4.02, p < 0.05$, with no other effects, $F_s < 1$. We compared the effect of immigration through 2-way ANOVAs (language group \times immigration status) on the 2 age variables. The monolingual group included 35 immigrants and 74 nonimmigrants, and the bilingual group contained 81 immigrants and 21 nonimmigrants. For both onset age, $F_{1,205} = 16.39, p < 0.0001$, and clinic age, $F_{1,207} = 12.07, p < 0.0007$, there were effects of language group but no effect of immigration status, $F_s < 1$.

There were no differences between language groups for MMSE scores, $F_{1,201} = 1.98$, NS, or gender, $F_{1,201} = 1.84$, NS, but monolingual subjects reported more formal schooling than bilingual subjects, $F_{1,205} = 9.08, p < 0.003$. This difference may reflect wartime circumstances in Europe at the time bilingual patients were teenagers. The discrepancy, however, again favors the monolingual subjects who received more formal education.

DISCUSSION Our interpretation of the present findings is that bilingualism is a cognitively demanding condition that contributes to cognitive reserve in much the same way as do other stimulating intellectual and social activities.^{1,6,9} These results replicate and extend our previous findings in several respects. The patients were all diagnosed with probable AD rather than a variety of dementias, and we collected more precise information about each patient's language, occupation, and immigration history. It should be noted that in both studies the levels of cognitive impairment of the 2 language groups were equivalent at the time of diagnosis; this fact reduces the likelihood that the result reflects some group difference in referral to the clinic.

Nonetheless, the findings should be treated cautiously given that the group comparison is cross-sectional; more definitive results would be obtained from a prospective study. The important question is whether bilingualism is truly the agent of change in these patients, or whether other factors associated with the bilingual group are responsible. Two candidates for such confounding factors are education and occupational status, but our present data show that the monolingual group has higher mean scores than the bilingual group on both of these variables, so any protective effect associated with more education or higher occupational level would work against our hypothesis. A third possibility is that immigrants may be more energetic than nonimmigrants, and therefore the bilingual effect is really an immigrant effect. In the present samples, 79% of the bilingual subjects but only 32% of the monolingual subjects were immigrants, supporting this possibility. An analysis taking immigration status into account within each

language group did not change our results, but caution is still warranted given the relatively small numbers involved.

The finding of a 4- to 5-year delay in the onset of symptoms of AD is dramatic. There are currently no pharmacologic interventions that have shown comparable effects. However, this estimate is in line with other studies showing a benefit of factors contributing to cognitive reserve. One review¹⁰ reports a reduction of 46% in the incidence of dementia associated with stimulating mental activities. We are not claiming that bilingualism in any way prevents AD or other dementias; the available evidence does suggest, however, that bilingualism postpones the onset of symptoms. The effects of this factor on the prevalence of AD in countries with high rates of bilingualism remain to be assessed.

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DISCLOSURE

Dr. Craik serves as a consultant for the Ontario Innovation Trust; serves on the editorial boards of *Psychology and Aging*, *Neuropsychology*, *Aging, Neuropsychology, and Cognition*, and *Memory*; receives royalties from the publication of *The Oxford Handbook of Memory* (Oxford University Press, 2000) and *Lifespan Cognition: Mechanisms of Change* (Oxford University Press, 2006); and receives research support from the Canadian Institutes of Health Research, the Alzheimer Society of Canada, Natural Sciences and Engineering Research Council of Canada. Dr. Bialystok serves on the editorial boards of *Bilingualism, Language and Cognition*; the *International Journal of Bilingualism*; and *Applied Psycholinguistics*; receives royalties from the publication of *Bilingualism in Development* (Cambridge University Press, 2001); and receives research support from the NIH (NICHD R01 HD052523 [PI]), the Canadian Institutes of Health Research, the Alzheimer Society of Canada, and the Natural Sciences and Engineering Research Council of Canada. Dr. Freedman serves on the editorial board of *Brain and Cognition*; receives royalties from the publication of *Clock-drawing: A Neuropsychological Analysis* (Oxford University Press, 1994); serves on scientific advisory boards for Pfizer Inc., Novartis, and Wyeth; and receives research support from Lundbeck Inc., the Saul A. Silverman Family Foundation as part of a Canada International Scientific Exchange Program, the Morris Kernzer Fund, the Canadian Institutes of Health Research, the Alzheimer Society of Canada, and the Ontario Mental Health Foundation.

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REFERENCES

1. Stern Y. Cognitive reserve. *Neuropsychologia* 2009;47:2015–2028.
2. Bialystok E. *Bilingualism in Development: Language, Literacy, and Cognition*. New York: Cambridge University Press; 2001.
3. Bialystok E, Craik FIM, Klein R, Viswanathan M. Bilingualism, aging, and cognitive control: evidence from the Simon task. *Psychol Aging* 2004;19:290–303.
4. Costa A, Hernandez M, Sebastian-Galles N. Bilingualism aids conflict resolution: evidence from the ANT task. *Cognition* 2008;106:59–86.

5. Bialystok E, Craik FIM, Freedman M. Bilingualism as a protection against the onset of symptoms of dementia. *Neuropsychologia* 2007;45:459–464.
6. Bennett DA, Wilson RS, Schneider JA, et al. Education modifies the relation of AD pathology to level of cognitive function in older persons. *Neurology* 2003;60:1909–1915.
7. Chertkow H, Whitehead V, Phillips N, Wolfson C, Atherton J, Bergman H. Multilingualism (but not always bilingualism) delays the onset of Alzheimer disease: evidence from a bilingual community. *Alzheimer Dis Assoc Disord* 2010;24:118–125.
8. McKhann G, Drachman DA, Folstein M, Katzman R, Price D, Stadlan EM. Clinical diagnosis of Alzheimer's disease: report of the NINCDS-ADRDA work group under the auspices of Department of Health and Human Services Task Force on Alzheimer's disease. *Neurology* 1984;34:939–944.
9. Wilson RS, Mendes De Leon CF, Barnes LL, et al. Participation in cognitively stimulating activities and risk of incident Alzheimer disease. *JAMA* 2002;287:742–748.
10. Valenzuela MJ, Sachdev P. Brain reserve and dementia: a systematic review. *Psychol Med* 2006;36:441–454.



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REFERENCES

1. French J, Gronseth G. Lost in a jungle of evidence: we need a compass. *Neurology* 2008;71:1634–1638.
2. Gronseth G, French J. Practice parameters and technology assessments: what they are, what they are not, and why you should care. *Neurology* 2008;71:1639–1643.
3. Gross RA, Johnston KC. Levels of evidence: taking *Neurology*[®] to the next level. *Neurology* 2009;72:8–10.

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U = Data inadequate or conflicting; given current knowledge, treatment (test, predictor) is unproven.

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